Amendments to the Specification:

Please replace the paragraph at page 1, from line 4 through line 13, with the following paragraph:

-- This application is a continuation-in-part of International Application No. PCT/US01/00565, which designated the United States and was filed on January 8, 2001, to be published in English, which is a continuation-in-part of Application Nos. 09/625,191, filed July 21, 2000, now abandoned; U.S.S.N. 09/543,371, filed April 4, 2000, now abandoned; and U.S.S.N. 09/479,118, filed January 7, 2000, now abandoned. U.S.S.N. 09/625,191 is in turn a Continuation-In-Part of U.S.S.N. 09/543,371, which, with U.S.S.N. 09/479,118, is a Continuation-In-Part of U.S.S.N. 09/335,224, filed June 17, 1999, now U.S. Patent No. 6,759,047, which in turn claims the benefit of U.S. provisional application 60/089,689, filed June 17, 1998 and U.S. provisional application 60/126,175, filed March 25, 1999. The entire teachings of all these applications are incorporated herein by reference. --

Please replace the paragraph at page 8, line 17 through page 10, line 2 with the following paragraph:

-- The invention also relates to an anti-angiogenic, isolated non-Goodpasture fragment of α3(IV) NC1 domain, which has one or more of the following characteristics: (a) the ability to bind $\alpha_v\beta_3$ integrin; (b) the ability to inhibit proliferation of endothelial cells; and (c) the ability to cause apoptosis of endothelial cells. The isolated non-Goodpasture fragment binds $\alpha_v\beta_3$ integrin by an RGD-independent mechanism, as described herein. Such an isolated fragment of the α3(IV)NC1 domain of Type IV collagen is described herein, and is designated "Tumstatin." "Tumstatin", as the term is used herein, comprises SEQ ID NO:10. In addition, another isolated non-Goodpasture fragment, designated herein as "Tum-1", or "Tumstatin N53" (SEQ ID NO:22), consists of the amino acid sequence of amino acid residue 54 to amino acid 244 of full-length Tumstatin (SEQ ID NO:10). Other isolated fragments disclosed herein include "Tum-2" (SEQ ID NO:23), "Tum-3" (SEQ ID NO:24), "Tum-4" (SEQ ID NO:25), and "Tum-5"

(SEQ ID NO:26), which consist of the amino acid sequence of residues 1 to 132 (Tum-2), residues 133 to 244 (Tum-3), residues 181 to 244 (Tum-4), and residues 54 to 132 (Tum-5) of full-length Tumstatin (SEQ ID NO:10), respectively. Peptide fragments are also disclosed herein, including "T1" (SEQ ID NO:27), "T2" (SEQ ID NO:28), "T3" (SEQ ID NO:29), "T4" (SEQ ID NO:30), "T5" (SEQ ID NO:31), "T6" (SEQ ID NO:32) and "T7" (SEQ ID NO:37), which consist of amino acid residues 1 to 19 (T1), 53 to 72 (T2), 68 to 87 (T3), 83 to 102 (T4), 98 to 116 (T5), 113 to 131 (T6) and 73 to 97 (T7), respectively, of full-length Tumstatin (SEQ ID NO:10). Yet another peptide fragment of full-length Tumstatin is designated herein as ["Tumstatin-45-132"] "Tumstatin-44-131" (SEQ ID NO:33) and consists of amino acid residues [45 to 132] 44 to 131 of full-length Tumstatin (SEQ ID NO:10). Another fragment of full-length Tumstatin is designated herein as "Tum-5-125-C-A" (SEQ ID NO:34), and consists of [Tumstatin-45-132] Tumstatin-44-131, where the cysteine at position 125 (of full-length Tumstatin) is mutated via site-directed mutagenesis to alanine. Fragments of Tumstatin which are reduced, *e.g.*, alkaline reduced, are also described herein to possess anti-angiogenic properties. Two other fragments are "Tumstatin 333" (SEQ ID NO:20) and "Tumstatin 334" (SEQ ID NO:21), which consist of residues 1 through 124 (Tumstatin 333) and residues 125 through 244 of full-length Tumstatin (SEQ ID NO:10). Other fragments of Tumstatin include T7-mutant (SEQ ID NO:38, methionine has been substituted for the leucine residue at position 77 of the full-length Tumstatin molecule, and isoleucine has been substituted for valine at position 81, and asparagine has been substituted for aspartic acid at position 83), T8 (SEQ ID NO:39, lysine has been substituted for the leucine residue at position 68 of the full-length Tumstatin molecule), T8-3 (SEQ ID NO:40, in which lysine has been substituted for the leucine residue at position 68 of the full-length Tumstatin molecule, and serine has been substituted for the cysteine residues at positions 79 and 85), TP3 (SEQ ID NO:41, in which lysine has been substituted for the phenylalanine residue at position 76 of the full-length Tumstatin molecule, and cysteine has been substituted for the aspartic acid at position 83), and P2 (SEQ ID NO:42, in which lysine has been substituted for the leucine residue at position 68 of the full-length

Tumstatin molecule, and [[and]] an aspartic acid has been substituted for the cysteine residues at positions 79 and 85). --

Please replace the paragraph at page 10, lines 3 through 20 with the following paragraph:

-- The invention also features an anti-tumor cell, isolated non-Goodpasture fragment of α3(IV) NC1 domain, which has one or more of the following characteristics: (a) the ability to bind $\alpha_v\beta_3$ integrin, (b) the ability to bind endothelial cells, (c) the ability to inhibit proliferation of tumor cells, and (d) the inability to inhibit proliferation of endothelial cells. The isolated non-Goodpasture fragment can bind $\alpha_v\beta_3$ integrin by an RGD-independent mechanism, as described herein. One isolated non-Goodpasture fragment comprises the amino acid sequence of amino acid residue [185] 184 to amino acid [203] 202 of full-length Tumstatin (SEQ ID NO:10). Another peptide fragment of full-length Tumstatin is designated herein as "T3," and consists of amino acid residues 68 to 87 of full-length Tumstatin (SEQ ID NO:10). Yet another peptide fragment of full-length Tumstatin is designated herein as ["Tumstatin-45-132,"] "Tumstatin-44-131," and consists of amino acid residues [45 to 132] 44 to 131 of full-length Tumstatin (SEQ ID NO:10). Another fragment of full-length Tumstatin is designated herein as "Tum-5-125-C-A" (SEQ ID NO:34), and consists of [Tumstatin-44-131] Tumstatin-44-131 (SEQ ID NO:33), where the cysteine at position 125 (of full-length Tumstatin) is mutated via site-directed mutagenesis to alanine. Fragments of Tumstatin which are reduced, *e.g.*, alkaline reduced, are also described herein to possess anti-angiogenic properties. Other fragments of Tumstatin include T7-mutant, T8, T8-3, TP3, and P2. --

Please replace the paragraph at page 30, lines 22 through 26 with the following paragraph:

-- Fig. 51 is a histogram showing the effect of *E. coli*-expressed [Tumstatin-45-132] Tumstatin-44-131 and Tum-5-125-C-A on progression of the cell cycle. The percentage of C-PAE cells in S phase (y-axis) is shown at hour 0 (control), and after

treatment by 0, 1, 10 and 20 μg/ml (x-axis) [Tumstatin-45-132] Tumstatin-44-131 (black bars) or Tum-5-125-C-A (white bars). The experiments were repeated three times. --

Please replace the paragraph at page 30, line 27 through page 31, line 8 with the following paragraph:

-- Figs. 52A, 52B, 52C and 52D are a set of four histograms showing the effects of [Tumstatin-45-132] Tumstatin-44-131 and Tum-5-125-C-A on cell viability. Fig. 52A shows cell viability as measured at OD_{562} (y-axis) in an MTT assay, for C-PAE cells treated with 0, 3, 6, 12, 25 and 50 μg/ml (x-axis) [Tumstatin-45-132] Tumstatin-44-131 (black bars) and [Tumstatin-45-132] Tumstatin-44-131 that was alkylated and reduced (white bars). Fig. 52B shows cell viability as measured at OD_{562} (y-axis) in an MTT assay, for C-PAE cells treated with 0, 3, 6, 12, 25 and 50 μg/ml (x-axis) Tum-5-125-C-A. Fig. 52C shows cell viability as measured at OD_{562} (y-axis) in an MTT assay, for PC-3 cells treated with 0, 3, 6, 12, 25 and 50 μg/ml (x-axis) [Tumstatin-45-132] Tumstatin-44-131. Fig. 52D shows cell viability as measured at OD_{562} (y-axis) in an MTT assay, for DU-145 cells treated with 0, 3, 6, 12, 25 and 50 μg/ml (x-axis) [Tumstatin-45-132] Tumstatin-44-131. --

Please replace the paragraph at page 31, lines 13 through 17 with the following paragraph:

-- Fig. 54 is a line graph showing the fractional tumor volume (y-axis) in terms of V/V_0 (mean tumor volume/initial tumor volume) at 0, 5, 10, 15 and 20 days (x-axis) of treatment with vehicle (control, □), 1 mg/kg [Tumstatin-45-132] Tumstatin-44-131 (♦), 1 mg/kg Tum-5-125-C-A (●), 20 mg/kg endostatin (○) and mini-pump administered [Tumstatin-45-132] Tumstatin-44-131 (1 mg/kg, Δ). --

Please replace the paragraph at page 46, line 22 through page 47, line 8 with the following paragraph:

-- Besides Tum-1, other Tumstatin deletion mutants were also created, including Tum-2, Tum-3 and Tum-4. These are also described in Example 35, below. Tum-1, as stated above, comprises the C-terminal 191 amino acids, and is lacking the N-terminal 53 amino acids. "Tumstatin 333" comprises the N-terminal amino acids 1 to 124 of Tumstatin. Tum-3 comprises the C-terminal 112 amino acids. Tum-4 comprises the C-terminal 64 amino acids, which includes amino acids [185-203] 184-202 (Han *et al.*, 1997, *J. Biol. Chem.* 272:20395-401). The region of amino acids [54 to 132] 53-131 of full-length Tumstatin was designated Tum-5. An extended version of Tum-5, designated herein as ["Tumstatin-45-132"] "Tumstatin-44-131", was created to increase the expression and solubility of Tum-5. ["Tumstatin-45-132"] "Tumstatin-44-131" consists of Tum-5, with an extension at the N-terminal end of an additional nine amino acids. In addition, a mutant of ["Tumstatin-45-132"] "Tumstatin-44-131" was created, designated herein as "Tum-5-125-C-A". This mutant consists of the sequence of ["Tumstatin-45-132"] "Tumstatin-44-131", where the cysteine at position 125 (of full-length Tumstatin) is mutated via site-directed mutagenesis to alanine. Further deletion mutants were made of Tum-5, which comprised T1 and a set of partially overlapping peptides (T2, T3, T4, T5 and T6). --

Please replace the table at page 47, line 11 through page 48, line 27 with the table below, which is marked up by way of bracketing and DOUBLE underlining to show the changes relative to the previous version of the table:

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Protein	Residues	Size	SEQ ID NO:
Tumstatin (full-length)	1 244	244	10
Tumstatin 333	1 124	124	20
Tumstatin 334	125 244	120	21
Tum-1 (Tumstatin N53)	54 244	191	22
Tum-2	1 132	132	23
Tum-3	133 244	112	24
Tum-4	181 244	64	25
Tum-5	54 132	79	26
T1	1 19	19	27
T2	53 72	20	28
T3	68 87	20	29
T4	83 102	20	30
T5	98 116	19	31
T6	[113 131] 113-131	19	32
Tumstatin-44-131 [45-132]	[45] 44 [132] 131	88	33
Tum-5-125-C-A	[45] 44 [132] 131[1]	88	34
T7	73 97	25	37
T7-mutant	73 97[2]	25	38
T8	68 94[3]	27	39
T8-3	68 94[4]	27	40
TP3	76 94[5]	19	41
P2	68 94[6]	27	42

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Please replace the paragraph at page 50, lines 24 through 29 with the following paragraph:

-- A mutant of [Tumstatin-45-132] Tumstatin-44-131 was created, Tum-5-125-C-A, in which the cysteine at residue number 125 (in the full-length molecule) is mutated to alanine. This mutation exhibits enhanced protein expression, and the molecule possesses anti-angiogenic properties equivalent to [Tumstatin-45-132] Tumstatin-44-131, with the exception of inhibition of tumor growth in mouse xenograft studies, where the mutant actually inhibited tumor growth more strongly than [Tumstatin-45-132] Tumstatin-44-131. --

Please replace the paragraph at page 68, lines 1 through 7 with the following paragraph:

-- The invention contemplates mutants of the proteins and peptides disclosed herein, where the mutation(s) do not substantially alter the activity of the protein or peptide, that is the mutations are effectively "silent" mutations. One such mutant, Tum-5-125-C-A, is presented herein, in which the cysteine at the 125th residue (of the full-length Tumstatin molecule) has been mutated from cysteine to alanine. This mutation prevents a disulfide bond from being formed at that residue, yet Tum-5-125-C-A retains the full activity of its parent molecule [Tumstatin-45-132] Tumstatin-44-131. --

Please replace the heading at page 156, line 6 with the heading below, which is marked -- Example 42. Expression and Purification of [Tumstatin-45-132] Tumstatin-44-131 and Tum-5-125-C-A. --

Please replace the paragraph at page 156, lines 19 through 22 with the following paragraph:

-- Tum-5-125-C-A (SEQ ID NO:34) was made by site-directed mutagenesis of residue 125 (of full-length Tumstatin) from cysteine to alanine, to enhance secretion of [Tumstatin-45-132] Tumstatin-44-131. It was expressed in *E. coli*, and was detected at

the same molecular weight size with western blotting using anti-polyhistidine tag antibody. --

Please replace the paragraph at page 156, line 23 through page 157, line 11 with the following paragraph:

-- Goodpasture syndrome is an autoimmune disease characterized by pulmonary hemorrhage and/or rapidly progressing glomerulonephritis, which are caused by the disruption of glomerular and alveolar basement membranes through immune injury associated with autoantibody activity against α3(IV)NC1. Recently, the most probable disease-related pathogenic epitope was identified in the N-terminal portion (Kalluri, R. *et al.*, 1996, *J. Biol. Chem.* 271:9062-8; Hellmark, T. *et al.*, 1999, *Kidney Int.* 55:938-44), and was then further confined within the N-terminal 40 amino acids (Hellmark, T. *et al.*, 1999, *J. Biol. Chem* 274:25862-8; Netzer, K.O. *et al.*, 1999, *J. Biol. Chem.* 274:11267-74). The N-terminal [Tumstatin-45-132] Tumstatin-44-131 consists of residues [45-132] 44-131 of Tumstatin, which is outside of the Goodpasture autoepitope. To further confirm that [Tumstatin-45-132] Tumstatin-44-131 would not be detected by Goodpasture autoantibody, antisera from patients with Goodpasture was used for western blotting. This antisera detected 293 cell-expressed full-length Tumstatin with high sensitivity, but failed to detect either *E. coli*-expressed [Tumstatin-45-132] Tumstatin-44-131 and *Pichia*-expressed Tum-5-125-C-A. This shows that [Tumstatin-45-132] Tumstatin-44-131 and Tum-5-125-C-A do not contain the Goodpasture autoepitope, and excludes the possibility that these recombinant proteins induce this autoimmune disorder upon administration in humans. --

Please replace the heading at page 157, line 12 with the following heading:

--Example 43. Activities of [Tumstatin-45-132] Tumstatin-44-131 and Tum-5-125-C-A. --

Please replace the paragraph at page 158, lines 3 through 13 with the following paragraph:

-- The effect of [Tumstatin-45-132] Tumstatin-44-131 and Tum-5-125-C-A on the cell cycle were assayed similarly to Example 4 above. Briefly, C-PAE cells were growth arrested by contact inhibition for 48 hours. The cells, at 10^5 cells per well, were then harvested and plated into a 12-well plate coated with fibronectin in 5% FCS and either recombinant [Tumstatin-45-132] Tumstatin-44-131 or Tum-5-125-C-A. After 21 hours, the cells were harvested and fixed in 70% ice-cold ethanol. The fixed cells were rehydrated a room temperature for 30 minutes in PBS containing 2% FCS and 0.1% Tween-20, centrifuged and resuspended in 0.5 ml of the same buffer. RNase (5 μg/ml) digestion was done at 37°C for one hour, followed by staining with propidium iodide (5 μg/ml). The cells were then counted using an EPICS XL-MCL flow cytometer (Beckman-Coulter Instruments, Fullerton, California, USA). --

Please replace the paragraph at page 159, lines 15 through 25 with the following paragraph:

-- Fig. 51 is a histogram showing G_1 arrest of proliferating endothelial cells. In the growth-arrested, contact-inhibited cells, 5.8% of the cells were in S phase at 0 hour. When the cells were stimulated with 5% FCS for 21 hours, there was a 3.7-fold increase in the percentage of cells in S phase, to 21.5%. Treatment with [Tumstatin-45-132] Tumstatin-44-131 decreased the percentage of cells in S phase to 6.0%. This effect was dose-dependent, with the percentage of cells in S phase being 19.3% at 1 μg/ml [Tumstatin-45-132] Tumstatin-44-131, and 11.3% at 10 μg/ml [Tumstatin-45-132] Tumstatin-44-131. In another experiment, the percentage of cells in G_0/G_1 phase was lowest in the 5% FCS-treated control group, and was elevated with treatment with [Tumstatin-45-132] Tumstatin-44-131. These results show that treatment with [Tumstatin-45-132] Tumstatin-44-131 causes cell cycle arrest in proliferating endothelial

cells. Treatment with Tum-5-125-C-A showed results equivalent to treatment with [Tumstatin-45-132] Tumstatin-44-131. --

Please replace the paragraph at page 159, lines 26 through page 160, line 6 with the following paragraph:

-- Figs. 52A, 52B, 52C and 52D are a set of four histograms showing the effects of [Tumstatin-45-132] Tumstatin-44-131 and Tum-5-125-C-A on cell viability. Fig. 52A shows cell viability as measured at OD_{562} (y-axis) in an MTT assay, for C-PAE cells treated with 0, 3, 6, 12, 25 and 50 μg/ml (x-axis) [Tumstatin-45-132] Tumstatin-44-131 (black bars) and [Tumstatin-45-132] Tumstatin-44-131 that was alkylated and reduced (white bars). [Tumstatin-45-132] Tumstatin-44-131 significantly decreased cell viability in a dose-dependent manner with an ED_{50} of 12 μg/ml. Reduced and alkylated Tumstatin and [Tumstatin-45-132] Tumstatin-44-131 exhibited effects similar to that of non-treated Tumstatin and [Tumstatin-45-132] Tumstatin-44-131 in decreasing cell viability of C-PAE cells. The anti-angiogenic effects of Tumstatin and [Tumstatin-45-132] Tumstatin-44-131 are therefore not dependent on their conformation as derived from disulfide bonds between cysteine residues. --

Please replace the paragraph at page 160, lines 7 through 10 with the following paragraph:

-- Tum-5-125-C-A exhibited effects in cell viability similar to those of [Tumstatin-45-132] Tumstatin-44-131, as shown in Fig. 52B. Fig. 52B shows cell viability as measured at OD_{562} (y-axis) in an MTT assay, for C-PAE cells treated with 0, 3, 6, 12, 25 and 50 μg/ml (x-axis) Tum-5-125-C-A. --

Please replace the paragraph at page 160, lines 11 through 17 with the following paragraph:

-- The effects of [Tumstatin-45-132] Tumstatin-44-131 and Tum-5-125-C-A on cell viability of C-PAE cells were not seen in control PC-3 and DU-145 cells, as shown in Figs. 52C and 52D. Fig. 52C shows cell viability as measured at OD_{562} (y-axis) in an MTT assay, for PC-3 cells treated with 0, 3, 6, 12, 25 and 50 μg/ml (x-axis) [Tumstatin-45-132] Tumstatin-44-131. Fig. 52D shows cell viability as measured at OD_{562} (y-axis) in an MTT assay, for DU-145 cells treated with 0, 3, 6, 12, 25 and 50 μg/ml (x-axis) [Tumstatin-45-132] Tumstatin-44-131. The activity of [Tumstatin-45-132] Tumstatin-44-131 is therefore specific to endothelial cells. --

Please replace the paragraph at page 164, line 15 through page 165, line 3 with the following paragraph:

-- [Tumstatin-45-132] Tumstatin-44-131 was also tested for its ability to suppress tumor growth. Male athymic nude NCRNU mice, of 5-6 weeks of age and about 25 g, were implanted with approximately 2 x 10^6 PC-3 (prostate cancer carcinoma) cells into the dorsal subcutis. The tumors were measured using Vernier calipers and the volume of the tumors calculated using the standard formula ($width^2$ x length x 0.52). The tumors were allowed to grow to about 50 mm^3, and animals were then pair-matched into groups of 6 mice. Initial doses of protein or vehicle (PBS, control) were given on the day of pair-matching (Day 1). [Tumstatin-45-132] Tumstatin-44-131, Tum-5-125-C-A, or human endostatin in sterile PBS was intraperitoneally injected daily b.i.d. at doses ranging from 1 to 20 mg/kg for 20 days. Control animals received injection of PBS vehicle. In one treatment, continuous subcutaneous delivery of [Tumstatin-45-132] Tumstatin-44-131 was done using surgically implanted Alzet mini-pumps. The mice were weighed twice weekly, and tumor measurements were taken, starting on Day 1. Estimated mean tumor volumes were calculated, and at Day 21, the mice were weighed, sacrificed, and their tumors excised and examined by light microscopy and CD31 immunostaining. The mean treated tumor weight was divided by the mean control tumor weight was subtracted from one, and expressed as a percentage to give the tumor growth inhibition for each group. --

Please replace the paragraph at page 165, lines 4 through 17 with the following paragraph:

-- The results are shown in Fig. 54, which is a line graph showing the fractional tumor volume (y-axis) in terms of V/V_0 (mean tumor volume/initial tumor volume) at 0, 5, 10, 15 and 20 days (x-axis) of treatment with vehicle (control, □), 1 mg/kg [Tumstatin-45-132] Tumstatin-44-131 (♦), 1 mg/kg Tum-5-125-C-A (●), 20 mg/kg endostatin (○) and mini-pump administered [Tumstatin-45-132] Tumstatin-44-131 (1 mg/kg, Δ). No toxicity from the protein treatments was seen, as judged by weight change. Both [Tumstatin-45-132] Tumstatin-44-131 and Tum-5-125-C-A significantly inhibited the growth of PC-3 cells. Human [Tumstatin-45-132] Tumstatin-44-131 at 1 mg/kg had a tumor growth inhibition of 74.1% ($p = 0.02$) and Tum-5-125-C-A had a tumor growth inhibition of 92.0% ($p = 0.001$), as compared to the vehicle-injected control group. Continuous delivery of [Tumstatin-45-132] Tumstatin-44-131 (1 mg/kg over 24 hours) via an Alzet mini-pump also showed significant tumor growth inhibition of 70.1% ($p = 0.03$). Endostatin delivered at a dose of 20 mg/kg (b.i.d., bolus injection) showed no significant tumor growth inhibition compared to the vehicle-treated control group. --

Please replace the paragraph at page 166, lines 5 through 10 with the following paragraph:

-- [Tumstatin-45-132] Tumstatin-44-131 intraperitoneal injection significantly inhibited microvessel density in PC-3 xenografts as compared to the vehicle-injected control group. The number of CD31-positive blood vessels per low power (40x) field was 6.33 ± 0.54 for [Tumstatin-45-132] Tumstatin-44-131 treatment, versus 9.44 ± 1.05 for the control ($p = 0.047$). Groups treated with Tum-5-125-C-A or the mini-pump-administered [Tumstatin-45-132] Tumstatin-44-131 showed similar decreases of mean vessel density. --

Please replace the paragraph at page 170, lines 15 through 25 with the following paragraph:

-- The potential capacity of tumstatin to inhibit protein synthesis in multiple endothelial cells was therefore explored. Tumstatin and its active subfragments, [Tumstatin-45-132] Tumstatin-44-131, T3 and T7 peptides were used. The amino acids [45-132] 44-131 of Tumstatin were expressed as recombinant [Tumstatin-45-132] Tumstatin-44-131 in *E. coli* as described above and in (Maeshima, Y. *et al.*, 2001, *J. Biol. Chem.* 276:15240-8). Human endostatin was produced in yeast as described in (Dhanabal, *et al.*, 1999, *Cancer Res.* 59:189-97). Only soluble protein with a low endotoxin level (less than 50 EU/mg) was used. T3 peptide, T7 peptide, consisting of residues 68-87 and 73-97 of tumstatin, respectively, and T7-mutant peptide (TMPFMFCNINNVCNFASRNDYSYWL; SEQ ID NO:38) were synthesized as described in (Maeshima, Y. *et al.*, 2000, *J. Biol. Chem.* 275:21340-8; Maeshima, Y. *et al.*, 2001, *J. Biol. Chem.* 276:31959-68). --